information on the proposed project or to obtain a copy of the data collection plans and instruments, contact Dr. Katherine A. McGlynn, Environmental Epidemiology Branch, DCEG, NCI, NIH, Executive Plaza South, Room 7060, 6120 Executive Boulevard, Bethesda, MD 20892–7234, or call non-toll-free number (301) 435–4918 or E-mail your request, including your address: *mcglynn@mail.nih.gov.*

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 30 days of the date of this publication.

Dated: February 14, 2002.

Reese L. Nichols,

NCI Project Clearance Liaison.

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7057; fax: 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Reagents to Examine the Expression and Function of CYP2J Subfamily P450s

Darryl Zeldin and Alyce Bradbury (NIEHS)

DHHS Reference No. E–033–02/0—Research tool

Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Cytochromes P450 catalyze the NADPH-dependent oxidation of arachidonic acid to various eicosanoids found in several species. The eicosanoids are biosynthesized in numerous tissues including pancreas, intestine, kidney, heart, and lung where they are involved in many different biological activities.

The NIH announces cloned cDNAs for several different CYP2J subfamily members and specific peptide-based antibodies to the P450 proteins. The reagents available for licensing include: human CYP2J2 cDNA, rat CYP2J3 cDNA, mouse CYP2J5 cDNA, mouse CYP2J9 cDNA, anti-CYP2J2rec, anti-CYP2J2pep2, anti-CYP2J9pep2, anti-CYP2J5pep, anti-CYP2J6pep, and insect cell microsomes expressing catalytically active CYP2J2. These reagents can be used to examine the expression of the CYP2J subfamily at the RNA and protein level and can be used to screen drugs for possible metabolism by the CYP2J2 subfamily P450s and/or to identify endogenous substrates for the enzyme. The recombinant protein may also be used to investigate cross-reactivity for other antibodies.

Polyclonal Antibody to Detect Human Membrane-Bound Protaglandin E Synthase

Dr. Thomas Eling et al. (NIEHS)

DHHS Reference No. E–032–02/0—Research Tool

Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Prostaglandin endoperoxide H_2 (PGH_2) is formed from arachidonic acid by the action of cyclooxygenases (cox)-1 or -2. Human prostaglandin E synthase (PGES) is a member of a protein superfamily consisting of membrane-associated proteins involved in eicosanoid and glutathione metabolism. PGE_2 a specific prostaglandin, is formed from PGH_2 by PGES and is then further metabolized into various eicosanoids. It has been reported that the membrane-bound mPGES is linked to cox-2 protein, which may be induced by proinflammatory cytokines such as IL–1β at sites of inflammation.

The NIH announces a polyclonal antibody capable of detecting human mPGES. It is anticipated that the use of this antibody in western analysis, immunostaining and immunoprecipitation studies will aid researchers in understanding prostaglandin creation and could eventually lead to the development of new anti-inflammatory agents.

Amyloid Beta is a Ligand for FPR Class Receptors

Dr. Ji Ming Wang et al. (NCI)

DHHS Reference No. E–336–01/0 filed 26 Oct 2001

Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Alzheimer's disease is the most important dementing illness in the United States because of its high prevalence. Five to ten percent of the United States population 65 years and older are afflicted with the disease. In 1990 there were approximately 4 million individuals with Alzheimer's, and this number is expected to reach 14 million by the year 2050. It is the fourth leading cause of death for adults, resulting in more than 100,000 deaths annually. Amyloid beta has been identified as playing an important role in the neurodegeneration of Alzheimer's disease. However, the mechanism by which this occurred was unknown, but has been postulated to be either direct or indirect through an induction of inflammatory responses.

The NIH announces the identification of the 7-transmembrane, G-protein-coupled receptor, FPRL–1, in the cellular uptake and fibrillar aggregation of amyloid ββ (Aββ) peptides. The Aββ peptides use the FPRL–1 receptor to attract and activate human monocytes and mouse microglial cells (publications referenced below), and have been identified as a principal component of the amyloid plaques associated with Alzheimer's disease. In addition, the known anti-inflammatory drug, Colchicine, has been shown to inhibit the FPRL1 activation by amyloid and the internalization of FPRL1/amyloid beta complexes.

This research has been published in Tiffany et al., ''Amyloid-beta induces chemotaxis and oxidant stress by acting at formylpeptide receptor 2 (FPR2), a G protein-coupled receptor expressed in phagocytes and brain'', J. Biol. Chem. 276(26):23645–52, 2001, and Cui et al., ''Bacterial lipopolysaccharide selectively up-regulates the function of the chemotactic peptide receptor FPR2 in murine microglial cells'', J. Immunol.168: 434–442, 2002.

System for in vivo Site-Directed Mutagenesis Using Oligonucleotides

Dr. Francesca Storici et al. (NIEHS)

DHHS Reference No. E–204–01/0 filed 27 Jul 2001

Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Through the use of molecular techniques to induce mutagenesis, along

with genetic functionality data, a large body of information is now available to characterize eukaryotic genomes. Yet with all the advancements seen, the techniques used have been unable to produce clean sequence modifications that contain no heterologous material and are flexible and easy to use.

The NIH announces a new technology wherein unpurified oligonucleotides can be used to create in vivo specific mutations that do not retain heterologous material following mutagenesis. This technology is versatile in that it will allow for site-specific mutagenesis as well as random mutagenesis within a localized area and is applicable to all organisms where homologous recombination is or can be performed. The technology allows for the generation of mutated products in vivo that contain only the desired mutation and can be used in multiple rounds of specific or random changes of up to 200 base pairs.

Fluorescent Magnesium Indicators

Drs. Robert E. London, Pieter Otten, and Louis A. Levy (NIEHS)

Serial No. 60/191,862 filed 24 Mar 2000 and Serial No. 09/816,638 filed on 23 Mar 2001

Licensing Contact: Pradeep Ghosh; 301/496–7736 ext. 211; e-mail: ghoshp@od.nih.gov

Magnesium is essential to many physiochemical processes and plays a central role in the biochemistry of all cells. Many epidemiological studies have established close association between plasma magnesium levels and various diseases including ischaemic heart disease, hypertension, atherosclerosis, osteoporosis, neurological disorders and other chronic illnesses. However, methods and tools to measure selectively ionized magnesium levels in cell preparations or in the body with accuracy and reliability are still lacking in the market today. The present invention pertains to analytical elements and methods for the selective determination of magnesium. In particular, the present invention relates to carboxy-quinolizones and their use as magnesium indicators. Thus, the present invention provides novel fluorescent indicators that are selective for $Mg2^{+}$. This invention utilizes fluorescence spectroscopy as a tool in monitoring intracellular or extracellular levels of magnesium. This is a non-invasive approach in which ion levels or ion fluxes induced by extra-cellular stimuli that can be monitored in real time. Current approaches used to measure devices to measure ionized intracellular magnesium in the body generally involve magnetic resonance spectroscopy to analyze intracellular ATP (adenosine triphosphate) signals. This approach is extremely expensive and subject to very poor accuracy. Unlike other methods and indicators, the composition and methods of this invention provide compounds with significantly increased abilities to accurately measure intracellular and extracellular $Mg2^{+}$ levels in a wide variety of cells. Further, an extended application of this invention relates to the monitoring of the effects of drugs, medicines or toxins that alter the intracellular magnesium levels via changes in cellular ATP levels.

Novel Anti-Thrombin Peptide From the Salivary Gland of Anopheles albimanus

Jesus G. Valenzuela, Jose Ribeiro, Ivo Francischetti (NIAID)

Serial No. 60/141,423 filed 29 Jun 1999 and PCT/US00/18078 filed 29 Jun 2000

Licensing Contact: Pradeep Ghosh; 301/496–7736 ext. 211; *e-mail: ghoshp@od.nih.gov*

Currently, there exists a need for effective bio-pharmacogenic inhibitors that can inhibit clot formation and platelet aggregation without lethal side effects. Blood clot formation resulting from platelet aggregation and chemical release may lead to several fatal vascular diseases such as myocardial infarction, strokes, pulmonary embolism, deep vein thrombosis, peripheral arterial occlusion and other cardiovascular thromboses. This invention pertains to the isolation and sequencing of an anticoagulant inhibitor. In particular, the invention describes the nucleic acid and amino acid sequences of anti-thrombin peptide anophelin, isolated from the salivary glands of the mosquito Anopheles albimanus. Alpha-thrombin has been reported to play an important role in the platelet dependent arterial thrombus formation leading to several life-threatening vascular diseases including myocardial infarction and strokes. The mosquito salivary anophelin described in this invention, referenced in Valenzuela et. al. Biochemistry. 1999 Aug 24;38(34):11209–15, is a novel, specific, tight-binding and effective inhibitor of alpha-thrombin. Biochemically, anophelin is a 6.5 kDa peptide that is easily synthesized, has no similarity to hirudin, and has no cysteines. The interaction of anophelin with anti-thrombin inhibits platelet aggregation and blood clotting. The current invention may be effectively administered in subjects, including humans, to inhibit alpha-thrombin activity by inhibiting platelet aggregation.

Identification of Compounds That Potentiate the Activity of Muscarinic Potassium Channels

David L. Armstrong and Desuo Wang (NIEHS)

DHHS Reference Nos. E–265–98/0 filed 30 Nov 2000 and E–265–98/1 filed 29 Nov 2001

Licensing Contact: Pradeep Ghosh; 301/496–7736 ext. 211; e-mail: ghoshp@od.nih.gov

Heart disease is one of the major causes of mortality in developed and developing countries. Potassium channel proteins regulate the excitability of heart muscle, and drugs that open potassium channels have been useful in treating human disease. The present invention describes a novel and G-protein independent mechanism for selectively stimulating muscarinic potassium channels (KIR 3.1/3.4 or KAch). KAch channels are a specific heteromeric class of potassium channels that regulate the excitability of atrial and modal monocytes in the heart in response to muscarinic receptor stimulation. Specifically, the present invention relates to compounds that potentiate the activity of muscarinic potassium channels in mammalian atrial monocytes and can treat cardiac disease. The present invention provides a novel mechanism for selectively stimulating KAch channels with tetraethylammonium (Wang & Armstrong 2000 J. Physiol. 529, 699–705. New drugs that selectively target the TEA site in the potassium channel without blocking other potassium channels may be able to relax the heart. Because TEA has been shown to enhance basal potassium channel activity without blocking or potentiating muscarinic stimulation, the danger of stopping the heart by targeting this site is minimized. In addition, because KACh channels are expressed primarily in atrial and nodal myocytes, the action potential duration would be shortened selectively in atrial and modal monocytes leading to slower pacemaker initiation and impulse condition without reducing ventricular force. Thus, identification of new drugs that target the TEA-site reported in this invention could have great market potential.

Dated: February 12, 2002.

Jack Spiegel,

Director, Division of Technology, Development and Transfer, Office of Technology Transfer, National Institutes of Health.

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